The contribution of mechanical power to ventilation-induced lung injury in the face of changing tidal volume

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Video Abstract

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Abstract

For patients in need of breathing assistance, mechanical ventilation can save lives. But it can also worsen or even initiate lung injury, which could prove fatal. Although excess tidal volume is associated with ventilator-induced lung injury, no prior reports address the effects produced by the combination of tidal volume and mechanical power – the energy transferred from a ventilator to the lungs as a function of time. To answer this question, an international research team looked at varying combinations of tidal volume, respiratory rate, and mechanical power to determine how each contributes to injury. Their results suggest that mechanical power plays a larger role than previously thought. The team randomized 32 Wistar rats with experimental mild acute respiratory distress syndrome to receive either low- or high-power mechanical ventilation, in combination with low or high tidal volume. In the low-power groups, the respiratory rate was adjusted to maintain normocapnia. In the high-power groups, the rate was adjusted to increase the mechanical power by threefold. Arterial blood gases and lung mechanics were measured at baseline and after two hours of ventilation. Alveolar damage and inflammatory biomarkers were also assessed. The results showed that the combination of high tidal volume and high mechanical power caused the greatest level of injury. Moreover, reducing the tidal volume did not fully protect against this injury. Markers of damage and inflammation were observed in the high-power groups even when a low tidal volume was maintained. These findings suggest that tidal volume and mechanical power independently contribute to ventilator-induced lung injury. However, although mechanical power was an important exacerbating factor, high tidal volume was still the main determinant of injury. For example, an increase in tidal volume more strongly triggered the pro-inflammatory response than an increase in mechanical power based on markers of inflammation, alveolar stretch, and alveolar epithelial and endothelial cell damage. High tidal volume was also more strongly associated with histologic evidence of alveolar damage as well as signs of mechanical stress and strain. Overall, although both factors contributed to damage, these findings suggest that the combination of low tidal volume and low mechanical power is most likely to protect against ventilator-induced lung injury.